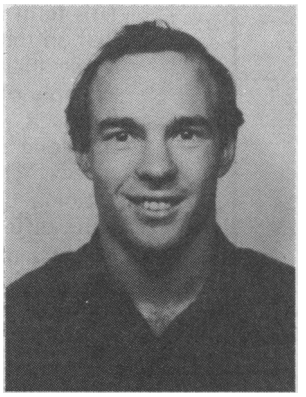
Runners, London Marathon, 1984 *By courtesy of All-Sport Photographic Ltd.*

C. Williams



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THE METABOLIC CHALLENGE OF THE MARATHON

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Key words: Marathon, Exercise, Endurance, Diet, Training.

INTRODUCTION

The energy expenditure required to complete the 42.2 km (26.2 miles) is almost equivalent to a marathon runner's daily energy intake. However only the stored carbohydrates and fat from the diet contribute to the fuel needs of the muscles during this endurance activity. These two fuels are degraded by oxidative metabolism within the muscle cells to supply the adenosine triphosphate (ATP) necessary for contractile activity. If the marathon was not a race, then the metabolic challenge would be within the scope of the energy stores of any healthy individual. The fact that it is a race means that the real challenge to the human metabolism is the need to sustain a high rate of energy expenditure for a prolonged period of time. When viewed in this light the weakness in human metabolism is that high rates of energy expenditure require a larger contribution from the body's limited carbohydrate stores than it does from the abundant supply of fat. This weakness in the system occurs because the skeletal muscles are unable to metabolise the available fatty acids at a rate which is fast enough to match the rate at which ATP is being used by the muscle cells. Training increases the aerobic capacity of the skeletal muscles and so not only improves the contribution of fatty acids to the energy needs of the working muscles but in so doing exerts a carbohydrate sparing effect. In this presentation these aspects of metabolism will be covered in slightly more detail as well as the dietary preparation necessary for successful participation in marathon running. But first it is helpful to consider some of the essential physiological responses to endurance exercise and to highlight the characteristics which distinguish those runners who are realistic contenders for the fastest times and those for whom finishing is in itself success.

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PHYSIOLOGICAL RESPONSES

As mentioned earlier, the energy required to complete a marathon is provided by the oxidative metabolism of carbohydrate and fatty acids. The cardio-respiratory system transports oxygen from the external environment to the internal environment of the muscle cells at a rate which is dictated by the metabolic demands of the working muscles. The oxygen uptake ($\dot{V}O_2$) of an individual increases as the exercise intensity increases until a point is reached above which there is no further increase in oxygen uptake. When the $\dot{V}O_2$ of a runner is plotted against increasing running speeds the linear relationship between $\dot{V}O_2$ and exercise intensity is clearly visible as is illustrated in Figure 1. The highest $\dot{V}O_2$ achieved is termed the maximum oxygen uptake ($\dot{V}O_2$ max) and it is an expression of the individual's physiological capacity for the transport and utilisation

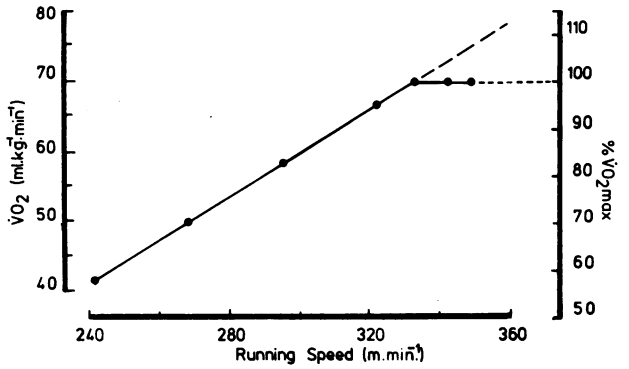

Fig. 1: A schematic representation of the relationship between oxygen uptake ($\dot{V}O_2$) and running speed. The $\dot{V}O_2$ values have also been expressed as % $\dot{V}O_2$ max.

of oxygen (Rowell, 1974). Elite marathon runners are, as a group, distinguished by their high $\dot{V}O_2$ max values which range from 70 to 80 ml.kg^{-1} min^{-1} (Davies and Thompson, 1979). When the $\dot{V}O_2$ of a runner, for a given speed, is expressed as a percentage of his or her $\dot{V}O_2$ max (%$\dot{V}O_2$ max) (Fig. 1) it reflects the relative exercise intensity experienced by that runner. For example, two runners can be running at the same speed and using the same amount of oxygen, but if they have different $\dot{V}O_2$ max values then they will experience different physiological responses to the run and so different levels of physiological stress. Cardiovascular, thermoregulatory and to a certain extent metabolic responses to exercise occur with respect to the relative exercise intensity (%$\dot{V}O_2$ max) rather than to the absolute exercise intensity (i.e. running speed or $\dot{V}O_2$ (Saltin and Hermansen, 1966; Hermansen et al, 1967; Rowell, 1974). This concept of relative exercise intensity has particular importance not only when considering potential racing speeds but also for selection of speeds which will produce the required training effect.

High speed sustained running requires a high rate of oxygen consumption, for example a 2 hr 18 min marathon (5:15 min/mile pace or 307 m.min^{-1}) demands a $\dot{V}O_2$ of about 58 to 60 ml.kg^{-1} min^{-1}. Therefore to be able to maintain this rate of oxygen consumption, an individual needs to have a $\dot{V}O_2$ max value which is higher than 60 ml.kg^{-1} min^{-1}. The elite marathon runners are able to tolerate running speeds which are equivalent to between 75% and 85% of their $\dot{V}O_2$ max values (Costill and Fox, 1969; Costill et al, 1971a; Davies and Thompson, 1979). This capacity to utilise a large %$\dot{V}O_2$ max appears to be one of the products of training because the recreational runner appears unable to tolerate such high relative exercise intensities (Maughan and Leiper, 1983). In addition to $\dot{V}O_2$ max values in excess of 70 ml.kg^{-1} min^{-1} many elite marathon runners possess what has been called "running economy". This term running economy is used to describe the fact that an individual is able to run at a certain speed with a lower than average $\dot{V}O_2$ for that particular speed. An example of differences in running economy is shown in Figure 2, where the $\dot{V}O_2$ values for a group of middle and long distance runners are plotted against running speeds. The runners M. D. and S. C., both of international class, have similar $\dot{V}O_2$ max values but have quite different rates of oxygen utilisation while running at the same speeds. Thus S. C. is an economical runner whereas M. D. is less than economical when compared with the average values for the group of top class runners used to illustrate this point. It is then, of considerable advantage to have a high $\dot{V}O_2$ max, and also to be an economical runner, in that high speed running can be tolerated because it represents a lower %$\dot{V}O_2$ max than it would for a less economical runner with a similar $\dot{V}O_2$ max. It also means that runners who do not have high $\dot{V}O_2$ max values, but who are economical runners can tolerate running speeds which are higher than what could be expected on the basis of their $\dot{V}O_2$ max values alone.

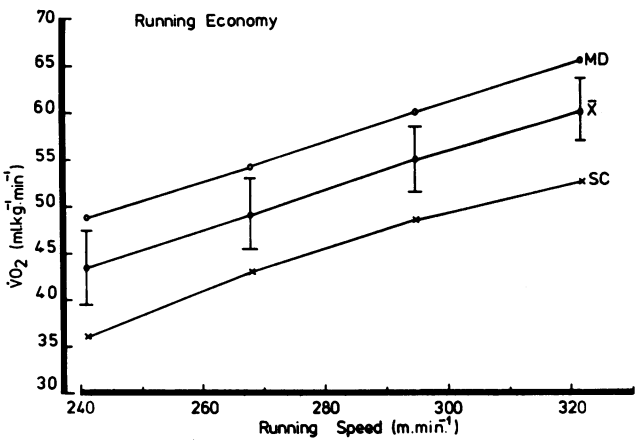

Fig. 2: The relationship between oxygen uptake ($\dot{V}O_2$) and running speed for a group of middle and long distance runners (n = 12). Included for comparison are the results of two international runners M. D. and S. C. Mean ± S.D. values (x) are shown for the group).

In addition to the characteristics described above, the top class marathon runners also have the capacity to utilise a large percentage of their $\dot{V}O_2$ max before developing a significant increase in blood lactic acid concentration (Sjodin and Jacobs, 1981). This quality reflects the large capacity of their skeletal muscles for the oxidative degradation of carbohydrate and fatty acids (Gollnick et al, 1972). This particular characteristic of elite marathon runners is partly the consequence of having skeletal muscles which are composed of mainly Type I fibres, which have a high capacity for oxidative metabolism and partly the product of training (Gollnick

et al, 1972). Endurance training increases the capacity of skeletal muscle for oxidative metabolism (Henriksson, 1977) and allows an individual to run at higher speeds, for a given blood lactic acid concentration, than was possible before training (Hurley et al, 1984).

Rather an attempt to assess at which running speed there is a significant increase in blood lactic acid concentration, some research workers have chosen to use blood lactic acid concentrations of 2 mmol.l^{-1} and 4 mmol.l^{-1} as reference values (Kindermann et al, 1979; Sjodin and Jacobs, 1981). An examination of the post-race blood lactic acid concentrations of marathon runners suggests that they are maintained around 2 mmol.l^{-1} (Costill and Fox, 1969). The running velocity equivalent to a blood lactic acid concentration of 2 mmol.l^{-1} (V2mM) provides a useful reference point for the description of the metabolic or training status of an endurance runner. For example, the average racing speeds of a group of elite marathon runners were found to correspond with treadmill speeds equivalent to a blood lactic acid concentration of 2 mmol.l^{-1} (Table I). In addition the V(2mM) corresponded to 79% $\dot{V}O_2$ max, which was the relative exercise intensity these marathon runners were able to tolerate during their best races. By way of comparison, the performance times for a group of recreation marathon runners who completed the 1984 London Marathon are shown in Table II, along with their V(2mM) values. Although their V(2mM) values were equivalent to 79% $\dot{V}O_2$ max, which was the same as the elite marathon runners, they were only able to tolerate an average speed which was 88.6% of their V(2mM) speed. Therefore even without an increase in their $\dot{V}O_2$ max values, further endurance training should eventually allow these recreational runners to be able to run at speeds equivalent to their V(2mM) values i.e. an increase of approximately 10% in their running speeds.

The energy expenditure during a marathon can be estimated from a knowledge of the runners performance time, his $\dot{V}O_2$ max, $\dot{V}O_2$ value for the average running speed, and an estimate of the energy equivalent per litre of oxygen consumed during the race. These physiological quantities are usually obtained from the results of tread-

TABLE I

Performance and physiological characteristics of elite male marathon runners (n = 5)

Runners	$\dot{V}O_2$ max ($ml.kg^{-1}\,min^{-1}$)	Marathon Time (min)	Marathon Speed ($m.min^{-1}$)	Marathon Speed (%$\dot{V}O_2$ max)	V(2mM) ($m.min^{-1}$)	V(2mM) (%$\dot{V}O_2$ max)	Marathon Speed (%V2mM)
P. G.	66.6	148.0	287	81	289	82	98.9
T. H.	73.8	137.5	307	82	304	81	100.8
M. M.	69.2	143.3	294	75	281	71	104.6
D. O.	67.7	146.0	289	83	303	86	95.5
N. S.	71.9	142.6	296	80	294	79	100.9
Mean	69.8	143.5	294	80.2	296	79.8	100.1
± S.D.	3.0	4.0	7.8	3.1	9.7	5.5	3.3

NB V2mM is the velocity ($m.min^{-1}$) equivalent to a blood lactate concentration of 2 mmol.l^{-1}

TABLE II

Performance and physiological characteristics of recreational marathon runners (n = 5)

Runner	$\dot{V}O_2$ max ($ml.kg^{-1}\,min^{-1}$)	Marathon Time (min)	Marathon Speed ($m.min^{-1}$)	Marathon Speed (%$\dot{V}O_2$ max)	V(2mM) ($m.min^{-1}$)	V(2mM) (%$\dot{V}O_2$ max)	Marathon Speed (%V2mM)
J. D.	57.8	193	218	71	244	79	89.7
E. F.	59.3	205	206	63	240	76	85.5
W. F.	58.5	215	196	76	208	78	94.4
J. K.	50.1	226	186	68	205	80	90.7
C. W.*	54.8	211	200	66	242	82	82.7
Mean	56.1	210	201	68.8	229	79.0	88.6
± S.D.	3.8	12.2	11.9	5.0	19.5	2.2	4.6

*Male runner. These 4 female and 1 male runners all completed the 1984 London Marathon

mill running experiments. Using these methods, Costill and Fox (1969) estimated that the energy expenditure of a group of marathon runners completing the race in an average time of 2.46 hr (147.6 mins) was 10.25 MJ (2542 kcal). This represents an average energy expenditure of approximately 70 KJ/min which, of course, reflects the exercise intensity. By way of comparison, the energy expenditures of an elite marathon runner (T. H.) and a recreational runner, (C. W.) both of whom completed the 1984 London Marathon, were calculated from the information of the type described above. The elite runner who completed the course in 2.29 hr (137.5 mins) used an estimated 10.4 MJ (2485 kcal) which is slightly more than his average daily energy intake (Table III). The recreational runner took over an hour longer to complete the same marathon course (Table II) and his estimated energy expenditure was 11.7 MJ (2798 kcal) which was also greater than his average daily energy intake of 10.45 MJ (2500 kcal). While the total energy expenditure of the recreational runner was greater than that of the elite runner, the rate of energy expenditure was, as would be expected, higher for the elite runner i.e. 75.5 KJ/min (18.07 kcal/min) viz 55 KJ/min (13.3 kcal/min). If both these individuals had been running at the same $\%\dot{V}O_2$ max, then the slower runners would have had the harder race, however the elite runner was able to utilise 81% $\dot{V}O_2$ max compared with only the 66% $\dot{V}O_2$ max used by the recreational runner and so the faster runner experienced the greater physiological stress.

TABLE III

Daily energy intakes (MJ, (Kcal)), and food composition of 10 elite male marathon runners.

Runner	MJ (kcal)	FAT		CHO		PROT	
		g	%	g	%	g	%
J. M.	13.3 (3173)	147	36	523	53	101	11
G. W.	13.3 (3176)	123	35	431	51	96	12
D. S.	13.4 i (3198)	139	39	363	43	93	12
R. S.	10.1 (2419)	83	31	350	54	74	12
D. O.	13.9 (3319)	130	35	416	47	132	16
M. M.	12.8 (3061)	103	30	483	59	69	9
P. B.	12.4 (2960)	82	25	449	57	86	12
T. H.	9.75 (2332)	80	31	326	52	85	15
K. L.	12.1 (2905)	104	32	426	55	85	12
H. Mc.	16.5 (3947)	151	34	529	50	100	10
Mean	12.8 (3049)	114	33	430	52	92	12
± S.D.	1.9 (456)	27	4	70	5	18	2

DIET

The energy intakes of a group of elite endurance athletes are shown in Table III. These values are not as high as one might expect for individuals who run at least 16 km (10 miles) each day. The average energy intake for this group is 12.8 MJ (3049 kcal), of which 52% is carbohydrate, 33% is fat and 12% is protein while the remaining 3% is accounted for by alcohol. These values are somewhat different from the values reported recently for middle-aged recreational runners who appeared to consume less carbohydrate and more fat than the elite runners (Blair et al, 1981). The consumption of greater amounts of carbohydrates by the elite runners compared to the recreational runners may be explained by their greater training distances and or the differences in ages between the two groups. While it is vitally important that the endurance runners cover their energy expenditure with their food intake, they cannot afford to increase their body weight unnecessarily. By way of comparison, collegiate wrestlers who also have a high daily energy expenditure as a result of their strength and power training programmes do not appear to have very high energy intakes. Their daily energy intakes are similar to those of endurance athletes, i.e. 13.55 MJ/day (3240 kcal) and may also be governed, to some extent, by the need to control their body weights (Houston et al, 1981).

Although the daily diet contains a range of different foods, the two main metabolic fuels are carbohydrate and fat. The simple and complex carbohydrates are eventually stored, after digestion and absorption, as a polymer of glucose called glycogen, in two main sites, namely the liver and muscle. The liver of an adult male, weighing approximately 1.8 kg contains about 90 grams of glycogen which is equivalent to 600 mmol glucosyl units per kg (wet weight) (Nisson and Hultman, 1973). Liver glycogen is mainly a reservoir of glucose which maintains the blood glucose concentration at an appropriate level to provide the brain and CNS with fuel for their metabolic activities. After an hour or more of prolonged low intensity exercise (i.e. 30% $\dot{V}O_2$ max) on a bicycle ergometer, blood glucose contributes significantly to carbohydrate metabolism in working muscles. However during running experiments, at speeds similar to those selected during marathon running, the contribution of blood glucose to overall carbohydrate metabolism may be as little as 12% (Hall et al, 1983). The ingestion of dilute glucose solutions (5%) may well prevent hypoglycaemia but it does not appear to improve endurance performance (Felig et al, 1982). Paradoxically, glucose ingested before prolonged submaximum exercise appears to contribute to, rather than delay, the early onset of fatigue. Costill and co-workers reported in 1977 that when a concentrated glucose solution was ingested within an hour of the start of prolonged exercise the result was an increase in carbohydrate metabolism during the subsequent exercise period and a reduction in endurance performance. The mechanism appears to involve an increase in plasma insulin concentration, following the ingestion of the glucose solution, which in turn exerts an antilipolytic effect. The decrease in the circulating fatty acid concentration following the increase in plasma insulin

denies the working muscles of its supply of this fuel and so the muscles use their limited glycogen stores at a more rapid rate. Recognising the consequences of attempting to take in "instant energy" in the form of glucose solutions or tablets immediately before exercise, the current recommendation is that an easily digestible carbohydrate meal should be consumed no later than 3 hours before the start of the marathon. The rate of fatty acid metabolism, by working muscles, is proportional to their plasma concentration (Armstrong et al, 1961). Elevated plasma fatty acid concentrations prior to exercise increases the amount of fatty acid metabolism during exercise an in so doing exerts a glycogen sparing effect (Rennie et al, 1976; Costill et al, 1977). Caffeine ingestion before exercise increases plasma fatty acid concentrations and the proportion of fat metabolism during subsequent submaximum exercise which results in an increase in endurance performance (Costill et al, 1978). Although the improvement in endurance performance may be the result of a glycogen sparing effect mediated by the lipolytic activity of caffeine there may be a more important effect on the CNS (Cadarette et al, 1983).

Fat is stored in the white adipose tissue cells in the form of triglycerides which are glycerol molecules to which are attached three long chain fatty acids. The free fatty acids and glycerol are mobilised when the glycerol are released into the blood stream where the fatty acids are transported in loose combination with albumin (Gollnick, 1977). Fatty acids are taken up by skeletal and cardiac muscle in proportion to their plasma concentrations. The fate of the fatty acids is either immediate metabolism within muscle mitochondria to produce energy in the usable form of ATP or the fatty acids are stored as intramuscular triglycerides. The oxidation of fatty acids in the mitochondria yields more ATP than the oxidation of an equivalent amount of carbohydrate. For example, the oxidation of 1 mmol of palmitic acid yields 129 mmol of ATP whereas the complete oxidation of 1 mmol of glucose yields 38 mmol of ATP.

In addition to the greater energy yield from the oxidation of fat, it also has the advantage that it is stored in the anhydrous state, whereas each gram of glycogen is stored with three to four grams of water (Olsen and Saltin, 1970). The disadvantage of fat as a fuel is that it can only release its potential energy after oxidative metabolism. Therefore when the energy provision from fat and carbohydrate per unit of oxygen consumed is considered, then carbohydrate yields more energy than fat. For example 1 litre of oxygen consumed would be equivalent to 19.7 KJ (4.70 kcal) when only fat is being oxidised whereas when carbohydrate is being oxidised the same oxygen consumption would yield 21 KJ (5.05 kcal). However rarely is one of these two fuels metabolised at the exclusion of the other and so the working muscles are always using both fuels in varying proportions depending on the exercise intensity and its duration. For example during exercise of gradually increasing intensity the aerobic metabolism of glycogen and fatty acids can cover the energy needs of the working muscles when the exercise is of low intensity. However when the exercise intensity increases the rate of ATP provision by oxidative metabolism may not be sufficiently fast to match the rate of ATP utilisation by the muscles. When this mismatching occurs the aerobic production of energy is complemented by a more rapid degradation of glycogen over the early part of the glycogenolytic pathway. As a consequence of this rapid glycogen degradation, 3 mmol of ATP molecules are produced in parallel with the aerobic production of 38 mmol of ATP from glycogen. Therefore although the additional rapid production of ATP occurs over a part of the glycogenolytic pathway (or more precisely, the Embden-Meyerhof pathway) which does not require oxygen and may therefore be regarded as anaerobic metabolism, there is still oxidative metabolism going on in the muscles. The rapid degradation of glycogen is accompanied by an increase in the concentration of lactic acid. The increased lactic acid concentration lowers the pH of the muscle cells and this is probably the single most important contributor to the onset of fatigue during high intensity exercise (Sahlin, 1978). In contrast fatigue during prolonged submaximum exercise is associated with a reduction or depletion of muscle glycogen rather than the accumulation of hydrogen ions (Ahlborg et al, 1967a, Hermansen et al, 1967; Gollnick et al, 1969).

CARBOHYDRATE LOADING

The importance of a diet rich in carbohydrate as part of the preparation for endurance performances has been established by the studies of Krogh and Lindhard (1920) and those of Christensen and Hansen (1939). In the latter study each subject exercised to exhaustion on a cycle ergometer at an exercise intensity equivalent to approximately 67% $\dot{V}O_2$ max. After three to four days on a low carbohydrate diet exercise time to exhaustion was 90 minutes whereas endurance time increased to 240 minutes after the same period of time on a high carbohydrate diet. The influence of prolonged high intensity exercise and subsequent consumption of a carbohydrate rich diet on muscle glycogen concentrations has been clearly shown by Bergström and Hultman (1966). In their now classical "one leg study" they showed that when one leg is exercised to exhuastion only the muscle glycogen stores in the exercised leg experiences glycogen depletion. Furthermore when a carbohydrate rich diet was consumed during the recovery period the glycogen stores were not only repleted but were almost double their pre-exercise values. This supercompensation phenomenon was confined to the muscles of the active leg and no such increase occurred in the inactive leg. In an attempt to exploit the supercompensation phenomenon Ahlborg and co-workers showed that when glycogen depletion

was followed by three days on a low carbohydrate diet and then three days on a high carbohydrate diet the supercompensation of muscle glycogen was most pronounced (Ahlborg et al, 1967b). This combination of exercise and diet has been shown to improve endurance performance during exercise on a cycle ergometer by between 38-50% (Ahlborg et al, 1967b; Bergström et al, 1967; Williams et al, 1976). Most of the experimental work on the influence of dietary procedures for increasing muscle glycogen concentrations have been carried out using cycle ergometers. Surprisingly little attention has been paid to the effectiveness of these dietary procedures on endurance capacity during running, even though they have been recommended as part of the preparation for participation in endurance competitions (Åstrand, 1967). Running to exhaustion does not appear to reduce the muscle glycogen stores to the same extent as during cycle ergometry and may not therefore be so closely associated with the development of fatigue (Karlsson and Saltin, 1971; Costill et al, 1971b). Nevertheless in one of the few studies on dietary carbohydrate loading and running performance Goforth and colleagues showed that at a running speed equivalent to 80% VO_2 max the improvement in endurance capacity was only 9% (Goforth et al, 1980). More recently improvements in treadmill running times of approximately 26% have been found using different forms of carbohydrate loading (Williams et al, 1984). In this study dietary carbohydrate loading was accomplished by supplementing the diets of the runners with either more of their habitual dietary carbohydrates, or simply by using confectionery products. These dietary manipulations were performed during the three days following a treadmill run to exhaustion at speeds equivalent to 70% VO_2 max. Running time to exhaustion was improved after only 72 hours of recovery with these dietary modifications. In this study one subject was able to complete the marathon distance, on the treadmill, after the 3 days on the high carbohydrate diet. An interesting observation was that during the run after the high carbohydrate diet, the subject appeared to use more carbohydrate than on the run following a mixed diet. The amount of carbohydrate used during the marathon run, which lasted 3.45 hr (207 min) was estimated from VO_2 values and respratory exchange ratios. Using this indirect method, the energy expenditure covered by carbohydrate metabolism was 8.68 MJ (2077 kcal) which represented 67% of the total energy expenditure of 12.96 MJ (3100 kcal) (Fig. 3). The estimated amount of carbohydrate used during the run was 542 g or approximately 2.6 g of carbohydrate per minute. This estimate is similar to the value reported by Hall et al (1983) who suggested that as much as 84% of the carbohydrate metabolism is the result of intra-muscular glycogen degradation. Using these values, the glycogen utilisation during the marathon run would have been of the order of 455 g.

While the available evidence supports the claims that

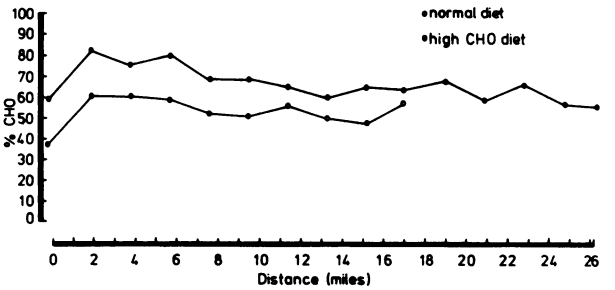

Fig. 3: An estimate of the % carbohydrate contributed to energy expenditure during two treadmill runs to exhaustion at 70% VO_2 max. The first run was performed without prior dietary modification (normal) while the second run was completed after three days on a high carbohydrate (CHO) diet.

dietary carbohydrate loading improves endurance performance there is only limited evidence to suggest that these procedures help improve performance times under race conditions. In the only reported study involving an endurance race (19 miles) glycogen-loading before the race improved the performance times of the subjects by an average of eight minutes or 5.6 per cent (Karlsson and Saltin, 1971). In this study, the speed of the subjects during the early part of the race did not increase after glycogen-loading, but they were able to sustain their optimal pace for longer during the latter part of the race. A closer examination of the results of this study reveals that the subjects could be divided into two groups, one group having higher VO_2 max values and faster race times than the other group. This first group was made up of experienced distance runners whereas the second group were active physical education students. The reduction in the running times for the 19 miles, were five minutes (3.2%) and 12 minutes (7.6%) for the first and second groups of runners respectively. The endurance runners had higher concentrations of muscle glycogen than the second group of subjects, and this may partly explain the differences in race times before and after the glycogen-loading procedures. The higher glycogen concentrations of the trained runners, without dietary modification, probably allowed them to sustain their optimal pace for more or less the entire race, hence the contribution of the dietary manipulation procedure was less effective for these individuals.

More recent studies reported by Sherman (1983) suggest glycogen supercompensation can be achieved without going through the low carbohydrate phase following prolonged exercise to exhaustion. However in the series of studies in which this procedure was used Sherman (1983) found that during a half-marathon time trial, the carbohydrate loaded runners did not improve on the running times they had recorded without prior carbohydrate loading. A possible explanation for this

negative finding is that the half-marathon distance is too short seriously to tax the muscle glycogen stores. An interesting observation reported by Sherman (1983) was that while there was no apparent benefit from carbohydrate loading for the half-marathon time trial, those individuals with the increased pre-race muscle glycogen concentration also used a greater amount of glycogen during the race. Thus the current recommendation is for the endurance athlete to taper his or her training during the 3 or 4 days before competition and to consume a diet rich in carbohydrate. The consumption of the rich carbohydrate diet can be achieved by reducing the protein content of the diet and making up the energy deficit with complex carbohydrates such as pasta, and rice.

TRAINING

As mentioned earlier, fatigue during prolonged exercise is associated with a reduction in muscle glycogen concentration below a certain critical level (Gollnick et al, 1975). Thus fatigue occurs in spite of the presence of an abundance of fatty acids. The failure to utilise fatty acids fully to cover energy metabolism when muscle glycogen concentrations are low appears to be associated with an inadequate mitochondrial capacity for oxidative metabolism. Thus the rate of oxidative degradation of fatty acids is too slow to match the rate at which ATP is being used for contractile activity by the muscles. Training, however increases the density of skeletal muscle mitochondria and this is accompanied by an increase in oxidative capacity (Holloszy and Booth, 1976). After training the endurance capacity is increased along with an increase in the proportion of fat oxidised to cover the energy needs of the working muscles (Henriksson, 1977). This increased oxidative metabolism of fat has a glycogen sparing effect which is obvious when an individual performs at the same exercise intensity as before training. The physiological responses to training are well known and include lower heart-rates, ventilation rates and blood lactate concentrations during submaximum exercise than were recorded before training (Saltin and Rowell, 1980). Rarely, however does the runner compete at the same speeds as were chosen before training. By and large the runner chooses a speed which may be regarded subjectively as representing "tolerable discomfort" or perceived simply as hard. Training expands the capacity for fat metabolism and so after training the runner may choose a speed which will still deplete the limited glycogen stores towards the end of the race, but the higher speed can be sustained because of an increased fatty acid and carbohydrate oxidation. A schematic diagram which attempts to illustrate these ideas is shown in Figure 4.

Training, therefore needs to be of sufficient intensity to stimulate the muscles to expand their aerobic capacity through an increase in mitochondria density. This is probably best achieved by the type of training used by

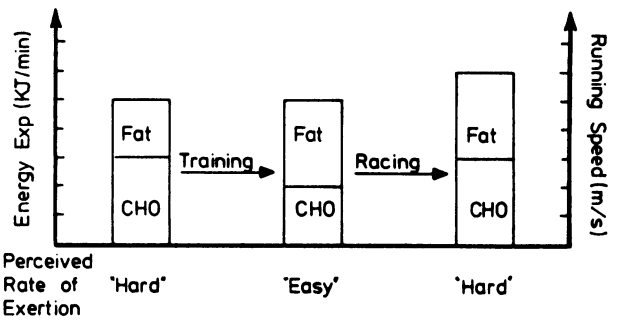

Fig. 4: A schematic representation of the relative contributions of fat and carbohydrate to energy metabolism during exercise before and after training and also at higher racing speed.

5,000 and 10,000 metre runners which usually involves sustained running at about 85-90% VO_2 max. The longer runs are necessary to tune the body to the cardiovascular and thermoregulatory demands of marathon running. In addition the metabolic preparation which occurs during the longer runs involves coping with the increased availability of fatty acids as a fuel along with educating the muscle fibre recruitment pattern so that all the Type I and Type IIA fibres can be enlisted to support locomotion.

Finally, the training-induced adaptations which are necessary for an individual to successfully meet the challenge of the marathon are as much a testimony to the plasticity of human metabolism as they are to the tenacity of the human spirit.

ACKNOWLEDGEMENTS

We are grateful for the help of Maria Nute and Steve Wootton during the various studies reported in this paper and also for the financial support from The Sports Council.

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